Research report

Are there attentional deficits in people putatively at risk for affective disorders?

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Abstract

Background: Schizophrenia is associated with attentional dysfunctions. In bipolar disorder, there is also evidence for sustained attention deficits. Therefore, we hypothesized that risk for bipolar disorder but not for unipolar depression might be associated with attentional abnormalities as well.

Method: Using the Hypomanic Personality Scale (HPS) and the Rrigidity Scale, we defined three groups: bipolar at-risk (n=42), unipolar at-risk (n=34), and control (n=37). All completed the d2 Test and the Continuous Performance Test (CPT).

Results: There was no evidence for overall attentional deficits in people at risk for affective disorders. However, reduced sensitivity, i.e., less discrimination between targets and nontargets, was observed in people at risk for bipolar disorders who also displayed schizotypy.

Limitations: We only looked at selective and sustained attention and did not assess other factors such as memory or executive functions. Additionally, the risk status was only defined by a psychometric indicator and did not include other approaches of defining risk (e.g., first-degree relatives).

Conclusions: Despite some limitations, our results support on one hand the idea that vulnerability for bipolar disorder can be associated with cognitive impairments, but they also highlight that this is not generally the case. Vulnerability for bipolar disorder and schizotypy might be correlated but are not the same.

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1. Introduction

Attentional processes are one of the core neuro-psychological functions involved in almost all performance. In the field of psychiatry, especially for schizophrenia, it was hypothesized that dysfunc-
tions in attentional processes are related to the pathophysiology of this condition, and many studies supported this idea by showing that attentional dysfunctions are found in relatives of patients with schizophrenia and schizotypy (e.g., Asarnow et al., 2002; Erlenmeyer-Kimling et al., 1993; Franke et al., 1994; Obiols et al., 1993, 1997). Therefore, the evidence is accumulating that aberrations in attentional processes are biobehavioral indicators for the vulnerability to schizophrenia (e.g., Cornblatt and Keilp, 1994; Erlenmeyer-Kimling and Cornblatt, 1992). Recently, more and more research is devoted to the question if such neuropsychological deficits are present in patients with unipolar and/or bipolar affective disorders (e.g., Albus et al., 1996; Bearden et al., 2001; Sax et al., 1995; Sevigny et al., 2003; Strauss et al., 1984). Typically, patients with affective disorders show impaired performance compared to healthy controls, and better performance than patients with schizophrenia but often no consistent differences between both patient groups are found (e.g., Albus et al., 1996; Mojtabai et al., 2000; Swann et al., 1999; Tabares-Seisdedos et al., 2003). One major question is if such neuropsychological deficits are a correlate, a consequence, or a precursor of affective symptoms. As a correlate, such deficits would just be symptoms of depression or mania and indicate differences in information processing (e.g., Murphy et al., 1999). If such deficits are still present during remission in euthymic patients but associated with characteristics of the disorder (e.g., presence or absence of psychotic symptoms in acute stages, number of episodes), then they would be consequences rather than causes and could indicate, for example, a degenerative process of the disorder itself (e.g., Albus et al., 1996; Martinez-Aran et al., 2004; van Gorp et al., 1998). If such deficits are, however, present during remission in euthymic patients but are unrelated with characteristics of the disorder or affected first-degree relatives show them as well, then the abnormalities could be risk or vulnerability factors for affective disorders (e.g., Clark et al., 2002; Gourovitch et al., 1999; Wilder-Willis et al., 2001; Zalla et al., 2004).

Another question that is often raised is if schizophrenia and affective disorders, especially bipolar disorder, are separate conditions or are better understood in terms of a continuum of symptoms. A lot of research has been looking at this issue lately, but the evidence supporting the thesis of an “unitary psychosis” is mixed (e.g., Crow, 1995; Torrey, 1999; Goldberg, 1999; Kromkamp et al., 2003). If the risks for both disorders are related, then the signs of vulnerability should be present in people at risk for schizophrenia and at risk for affective disorders. For example, Zalla et al. (2004) were able to show that first-degree relatives of patients with schizophrenia and those of patients with bipolar disorder demonstrated enhanced susceptibility to interference and reduced inhibition. This suggests that there is a shared vulnerability to both schizophrenia and bipolar disorder. As mentioned before, deficits in attentional processes were often documented as being associated with risk for schizophrenia, either defined biologically (e.g., children of patients; e.g., Erlenmeyer-Kimling et al., 1993; Franke et al., 1994) or psychometrically (e.g., Perceptual Aberration Scale; e.g., Kendler et al., 1991; Lenzenweger et al., 1991; Obiols et al., 1993).

To our knowledge, it has, however, never been investigated if the risk for affective disorders—psychometrically defined—is associated with attentional deficits. Because of the abovementioned discussion of the relation of schizophrenia and bipolar disorder, one might expect that the vulnerability for bipolar disorder is associated with similar neuropsychological deficits as the risk for schizophrenia. First evidence supports this hypothesis (e.g., Wilder-Willis et al., 2001). In the area of the psychometric high-risk approach, there is evidence that attentional dysfunctions are associated with schizotypy (e.g., Lenzenweger et al., 1991; Obiols et al., 1993), however, within the field of the psychometric high-risk research for affective disorders, there is a lack of empirical studies.

Searching for appropriate indicators for proneness to affective disorders, von Zerssen’s (1996) model seemed to be an interesting approach. He proposed two classes of premorbid personality as partial explanations of affective disorders: the Typus Melancholicus (TMEL), first described by Tellenbach (1961), predisposing to episodes of melancholia; and the Typus Manicus (TMAN), predisposing to the manic component of a bipolar disorder. Rigidity had been indicated by a number of studies to be a central
feature of TMEL (e.g., Lauer et al., 1997; Maier et al., 1992; Sauer et al., 1997). Although no longitudinal study has ever tested this hypothesis, rigidity or obsessive–compulsive traits are mentioned as possible risk factors for depressive symptoms. Because most studies based on von Zerssen’s ideas used the subscale “Rigidity” of the Munich Personality Test (MPT; von Zerssen et al., 1988), we decided to include this scale as a measure for proneness to depression.

As psychometric vulnerability indicator for the other pole of affective disorder, the TMAN (also related to Akiskal’s concept of hyperthymic temperament, Akiskal, 1996; Akiskal and Akiskal, 1992), we chose the Hypomanic Personality Scale (HPS; Eckblad and Chapman, 1986), although it has never been really linked to one specific theoretical model. High scorers have been described as “upbeat, gregarious, confident and energetic” (p. 216), needing habitually less sleep than other people, and disposed towards being irritable, rude, reckless, and irresponsible. Despite the atheoretical nature of the HPS, the evidence suggests that high scorers on the HPS demonstrate psychosocial impairment and are more likely to experience episodes of depression and/or (hypo-) mania (Eckblad and Chapman, 1986; Klein et al., 1996; Meyer and Hautzinger, 2003a). It has been shown that HPS is not just measuring neuroticism or extraversion (e.g., Meyer, 2002a) and that there is familial resemblance in HPS scores (Meyer and Hautzinger, 2003a). Most compelling is that the HPS predicted manic symptoms and the onset of bipolar disorders but not psychosis (Blechert and Meyer, in press; Kwapil et al., 2000).

Based on the abovementioned literature, we expected that people at risk for bipolar disorder, characterized by high HPS scores, would also display deficits in attentional functions—sustained attention and selective attention—as shown before for schizotypal persons, and there is some association between HPS and schizotypy as well (e.g., Allen et al., 1987; Meyer, 2002b). High HPS scorers might therefore also show higher schizotypy. Here, it is of interest if such deficits are related to schizotypy or vulnerability to bipolar disorders. However, this would be not the case for people prone to depression, defined by rigidity, because, first of all, depression is less often associated with psychotic symptoms than mania (e.g., Dilsaver et al., 1997; Goodwin and Jamison, 1990). Furthermore, vulnerability to depression seems not to be related to schizotypy (e.g., Allen et al., 1987). Additionally, no study seems to have found evidence for persistent attentional dysfunctions in depression (e.g., Lampe et al., 2004; Liu et al., 2002).

2. Method

2.1. Participants

Study participants were 6000 students who either still attended college or high school or went to vocational schools receiving a specific training for a job (e.g., cosmetology, mail services, banking, etc.). Subjects provided written consent prior to enrolling the study. All students completed the German version of the Hypomanic Personality Scale (HPS; Meyer, 2002a,b; Meyer and Hautzinger, 2003a), the subscale Rigidity of the Munich Personality Test (MPT; von Zerssen et al., 1988), the Magical Ideation Scale (MI; Eckblad and Chapman, 1983), and the Lie scale of the Eysenck Personality Inventory (EPI; Eysenck and Eysenck, 1964).

The questionnaires were completed in the class rooms. From this original sample of 6000 students, we selected three groups of students that corresponded to different vulnerability status for affective disorders based on their HPS and Rigidity scores (for details about the study, see Blechert and Meyer, in press): (1) Group with vulnerability for bipolar disorders (bipolar at-risk): students whose scores were in the upper 10% of the score distribution of the original cohort in the HPS, \( n = 58 \); (2) Group with vulnerability for unipolar depression (unipolar at-risk), \( n = 39 \): students whose score were in the upper 10% of the score distribution of the original cohort in the Rigidity Scale; (3) Control group (control): subjects who did not score 1/2 S.D. above the mean.

1 People were assigned to this group regardless of the scores in Rigidity for several reasons. First of all, bipolar disorder is always diagnosed if recurrent manic or hypomanic episodes are observed regardless of depressive episodes (APA, 1994). Furthermore, when we compared people scoring high on HPS with individuals scoring high on HPS and RIG, no significant differences emerged for schizotypy or attentional performance indices (available on request from the authors).
of the original sample in neither the HPS nor the Rigidity Scale, \( n=44 \). Non-native German students were not included in the sample. Additionally, individuals whose Infrequency score was 3 or higher (Chapman et al., 1984) and whose score on the Lie scale of the EPI was 5 or higher (Eysenck and Eysenck, 1964) were also not included in the sample. Finally, people belonging to one of these three groups defined above were interviewed with the Structured Clinical Interview for DSM-IV (SCID; First et al., 1995). Participants with current or past affective or psychotic disorder as determined by the SCID were also not included in the sample \( (n=28) \). This resulted in a sample of \( n=113 \) students (predominantly female with 67.3%). The mean age was 17.7 (S.D.=2.01, range: 15–23). Forty-eight percent \( (n=49) \) of the students had a high level of education \( (13 \text{ years of education, “Abitur”}) \), 16.7% \( (n=17) \) of students attended school for 10 years, representing an intermediate level of education \( (“\text{Mittlere Reife”}) \), and 35.3% of the students had either a low level of education \( (9 \text{ years, “Hauptschule”}) \) or not yet completed school at the time of the study. Descriptives for the three groups \( (\text{bipolar at-risk: } n=42, \text{ unipolar at-risk: } n=34, \text{ controls } n=37) \) are displayed in Table 1.

### Table 1

<table>
<thead>
<tr>
<th></th>
<th>Control ((n=37))</th>
<th>Unipolar at-risk group ((n=34))</th>
<th>Bipolar at-risk group ((n=42))</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean (S.D.)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>17.89 (2.00)</td>
<td>17.68 (2.06)</td>
<td>17.64 (2.02)</td>
</tr>
<tr>
<td><strong>HPS score</strong></td>
<td>17.25 (4.27)</td>
<td>16.06 (5.57)</td>
<td>34.02 (3.23)</td>
</tr>
<tr>
<td><strong>Rigidity score</strong></td>
<td>1.87 (1.06)</td>
<td>5.62 (0.65)</td>
<td>2.90 (1.99)</td>
</tr>
<tr>
<td><strong>Current symptoms(a)</strong></td>
<td>12.08 (2.12)</td>
<td>12.02 (2.48)</td>
<td>11.69 (2.47)</td>
</tr>
<tr>
<td><strong>Depressive</strong></td>
<td>12.05 (7.25)</td>
<td>12.85 (8.11)</td>
<td>16.90 (9.20)</td>
</tr>
<tr>
<td><strong>Hypomanic</strong></td>
<td>5.31 (2.61)</td>
<td>4.91 (2.48)</td>
<td>6.47 (3.29)</td>
</tr>
<tr>
<td><strong>Schizotypy(b)</strong></td>
<td>6.37 (3.51)</td>
<td>5.88 (3.38)</td>
<td>9.26 (4.47)</td>
</tr>
<tr>
<td><strong>IQ</strong></td>
<td>58.29 (5.45)</td>
<td>57.20 (6.35)</td>
<td>58.71 (5.57)</td>
</tr>
<tr>
<td><strong>d2</strong></td>
<td>178.1 (25.2)</td>
<td>181.3 (40.2)</td>
<td>180.8 (35.1)</td>
</tr>
<tr>
<td><strong>Errors (%)</strong></td>
<td>4.51 (3.15)</td>
<td>5.89 (4.65)</td>
<td>3.98 (2.67)</td>
</tr>
<tr>
<td><strong>CPT</strong></td>
<td>1.93 (0.79)</td>
<td>1.67 (0.88)</td>
<td>1.61 (0.78)</td>
</tr>
<tr>
<td><strong>ln beta</strong></td>
<td>2.21 (1.17)</td>
<td>1.54 (1.00)</td>
<td>1.84 (1.02)</td>
</tr>
</tbody>
</table>

The unadjusted means of the analysis of variance are displayed. Unipolar at-risk: High scores on the Rigidity Scale; Bipolar at-risk: High scores on the Hypomanic Personality Scale; IQ—general intelligence; d2—Selective attention task; KL—performance index of the d2; CPT—Continuous Performance Test; d’—sensitivity; In beta—reaction criteria (Grier, 1971).

\( a\) Current symptoms were assessed with the modified Center for Epidemiological Studies-Depression Scale.

\( b\) Schizotypy—Magical Ideation Scale.

#### 2.2. Self-report measures

The HPS includes 48 true–false items (e.g., “I am frequently so hyper that my friends kiddingly ask me what drug I’m taking”). The HPS has demonstrated good high internal consistency and test–retest reliability (Eckblad and Chapman, 1986; Meyer, 2002a,b; Meyer and Keller, 2003). The subscale Rigidity of the Munich Personality Test (MPT; von Zerssen et al., 1988) is also associated with risk for affective disorders (e.g., first-degree relatives of bipolar patients show such rigid or obsessive–compulsive traits; Maier et al., 1992, 1995; Lauer et al., 1997). Briefly, this scale is comprised of eight items assessing performance orientation, perfectionism, anacasm, and adherence to social norms (e.g., “Whenever I start something, I insist on doing it to perfection”). The Rigidity Scale has demonstrated good test–retest reliability and internal consistency (von Zerssen et al., 1988). The items of all the scales, including the control scales (Lie scale from the EPI and Infrequency Scale), were presented intermixed. As an indicator for schizotypy, we chose the Magical Ideation Scale (Eckblad and Chapman, 1983) that taps uncommon experiences and deviations in information...
processing and logical reasoning that is related with risk for psychosis (e.g., Kwapil et al., 1997). The English as well as the equivalent German scales have high internal consistency and are sufficiently reliable (e.g., Eckblad and Chapman, 1983; Meyer, 2002b).

The Center for Epidemiological Studies–Depression Scale (CES–D) and its extension (Radloff, 1977; Meyer and Hautzinger, 2003b) originally consists of 20 items assessing depressive symptoms. Responses to the items are rated using a four-point scale: “0” (rarely or none of the time), “1” (some or a little of the time), “2” (occasionally or a moderate amount of the time), and “3” (most of the time). For these four points, anchors are described specifying what it means to say “some or a little of the time”. Participants are asked how often they have experienced each of the symptoms during the previous week. For the assessment of hypomanic or manic symptoms, Meyer and Hautzinger (2001b; 2003b) included questions covering the criteria of the DSM-IV. The psychometric properties are good (e.g., Meyer and Hautzinger, 2001b, 2003b).

2.3. Cognitive functions

A degraded version of the Continuous Performance Test (CPT; Kathman et al., 1996; Nuechterlein et al., 1994) was used to assess vigilance/sustained attention to be comparable to other studies. As dependent measures, we chose the indices from signal detection theory: d’ (sensitivity), i.e., the ability to discriminate between the target and nontarget, and ln beta (response criterion), i.e., the assessment of how much perceptual evidence a person needs before reacting. Lower betas indicate a risky bias (Grier, 1971). On a computer monitor, a total of 480 trials are run at a rate of one per second. The numbers from 0 to 9 that appear are being partly masked (degraded). Reliability was reported to be good with 0.83 to 0.92 (Kathman et al., 1996; for review of CPT results and measure: Cornblatt and KeIlp, 1994; Nuechterlein et al., 1994).

As a measure of selective attention, we chose the d2 Test (Brickenkamp, 1994). It assesses attention via both speed and accuracy of performance. The task is to discriminate visually similar targets and nontargets. The target is the letter d if it is accompanied by two additional lines (e.g., on the top of the letter or on the bottom). The test is a paper-and-pencil test and has 14 rows each consisting of 47 items, i.e., letters. The letter d accompanied by one, three, or four lines is a nontarget, and the same holds true for the letter p regardless of the number of lines (see Fig. 1). The subject has 8 min to cross out all targets but is asked by the experimenter to stop working on the current row and continue with the next row every 20 s. The reliability of all measures is judged to be high (>90; Brickenkamp, 1994), and the test is widely used (e.g., Hoppner et al., 2003; Sauer et al., 2003; Seal, 2004).

To control for and estimate general intelligence, we used a shortened version of the “Leistungsprüfungssystem” (LPS; Horn, 1983) that is a paper-and-pencil test. Instead of the original 14 subtests requiring about 2 h of testing, the short version is equivalent to the longer version and contains only six tests: vocabulary, reasoning, word fluency, spatial orientation, flexibility of closure, and speed of closure (Sturm and Willmes, 1983).

2.4. Statistical procedure

The main hypotheses are tested by conducting analyses of variance for the dependent measures. In case of a significant main effect, analysis of covariance was run next to control for general intellectual functioning and self-reported mood symptoms. For post hoc comparisons, Scheffé tests were used. For comparisons between two groups, one-tailed p-values for t-tests were used if a specific hypothesis was tested.

3. Results

First, it seemed important to take a look at baseline characteristics of the three groups (Table 1). They did, as expected, differ regarding the HPS.
[\(F(2,113)=212.15, p<0.011\)] with only the bipolar at-risk group having significantly higher scores than the other two groups. Concerning Rigidity scores, we also found the expected difference, but this time all three groups differed significantly from each other [\(F(2,113)=66.78, p<0.001\)]. Groups did not differ on age [\(F(2,113)=0.17, \text{n.s.}\)], or global intelligence [LPS: \(F(2,113)=0.63, \text{n.s.}\)].

Looking at schizotypy, we found, as others, that individuals in the bipolar at-risk group had significantly higher MI scores than individuals in the unipolar at-risk or control group, with the latter two not being significantly different from each other [\(F(2,107)=8.28, p<0.001\)].

Although we excluded individuals with affective and psychotic disorders, we nevertheless assessed current mood symptoms. We found overall effects for both current subthreshold depressive [\(F(2,113)=3.88, p<0.05\)] and hypomaniac symptoms [\(F(2,113)=3.15, p<0.05\)]. However, post hoc comparisons with Scheffe tests revealed that the only significant difference was between control and bipolar at-risk group, with the latter one reporting more depressive symptoms (\(p<0.05\)). Correcting for multiple testing, current hypomanic symptoms did not differ between groups any more.

To determine the relations of risk group with neuropsychological data from the d2- and the CPT tests, separate analyses were conducted for the two tests. We first looked at the main concentration index (KL). The KL of the d2 Test reflects accurate performance corrected for mistakes. The groups, however, did not differ regarding KL [\(F(2,213)=0.09\)]. Table 1 displays all descriptives for attentional functioning. The percentage of errors (omissions and false alarms) showed a trend with the bipolar at-risk group, producing least errors, and the unipolar at-risk group, most errors [\(F(2,113)=2.84, p=0.06\)]. However, as current symptoms and intelligence could have significantly influence on the performance, we included self-reported depressive and hypomaniac symptoms as well as global intelligence (measured by the LPS) as covariates into the analysis. This ANCOVA yielded a significant effect only for LPS, \(F(1,103)=7.36, p<0.01\); the other covariates ADS_D and ADS_M did not reach significance (both \(F<1.0\)). After controlling for these variables, the group status did not approach significance any more [\(F(2,102)=1.89, p=0.16\)]. Therefore, we did not find any evidence that risk for affective disorder was related to problems with selective attention after adjusting for general intelligence.

Switching to sustained attention and the more widely used CPT, we analyzed the performance in a similar way. One-way ANOVAs first tested for the differences of the test results between the risk groups without taking into account current symptoms and intelligence. The latter was added to the analysis in an ANCOVA if significant differences were obtained. The sensitivity index \(d'\), indicating discrimination between targets and nontargets, did not differ between the three groups [\(F(2,113)=1.71, \text{n.s.}\)].

Because we hypothesized that only the bipolar at-risk group and the control group would significantly differ from each other, we also tested a planned contrast. Since the bipolar at-risk group had higher schizotypy scores and lower sensitivity, \(d'\), we decided to split this group in a bipolar at-risk group with schizotypy (MI=10, \(n=15\)) and without schizotypy (MI\(\leq 10, n=27\)) and separately compared these with the control group. While control and bipolar at-risk groups without schizotypy did not differ significantly from each other [\(t(62)=1.35, p=0.09; 1.93 (\text{S.D.}=0.79)\) vs. 1.66 (S.D.=0.84)], the schizotypal individuals in the bipolar at-risk group discriminated significantly worse between target and nontarget than controls [\(t(50)=1.90, p<0.05; 1.52 (\text{S.D.}=0.67)\) vs. 1.93 (S.D.=0.79), respectively].

Looking at the response criterion, In beta, a significant effect emerged [\(F(2,113)=3.55, p<0.05\)] for risk group. Post hoc Scheffe tests revealed that only the unipolar at-risk group significantly differed from the control group. Even after controlling for current symptoms [depression: \(F(1,103)=1.10, \text{n.s.}\); hypomania: \(F(1,103)=2.04, \text{n.s.}\)] and intelligence \(F(1,103)=0.29, \text{n.s.}\), the effect of the risk status remained significant [\(F(1,103)=3.59, p<0.05\)]. Compared to controls, the individuals in the unipolar at-risk group adopted a more risky strategy in reacting, i.e., reacted earlier thereby producing more false alarms (the correlation of the number of false alarms with In beta was \(-0.77, p<0.01\)).

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\(^2\) Similar analyses could not be done for people at risk for unipolar depression because only two individuals scored about the cutoff of 10 for the MI scale; this is also reflected in the significant difference between groups for schizotypy scores. Magical Ideation and Rigidity were unrelated in this sample (\(r=0.01, n=133\)).
Finally, we assessed the relationships between the selective and sustained attention by looking at their intercorrelation \((n=108)\). The sensitivity index of the CPT, \(d'\), only moderately correlated with the KL score of the d2 Test \((r=0.25, p<0.05)\) and slightly negatively with the error percentage \((r=-0.19, p<0.01)\). The reaction criterion, ln beta, of the CPT was unrelated to performance \((r=0.06)\) and error rate of the d2 Test \((r=-0.08)\). This pattern suggests that the two tests tap different aspects of attention.

### 4. Discussion

Based on the empirical evidence for attentional deficits in schizophrenia and affective disorders, especially bipolar disorder, we looked if such abnormalities in attentional processes are present before the onset of the disorder in people hypothesized at risk for bipolar disorder. We specifically looked at selective attention using the d2 Test and sustained attention using the well-established CPT. Overall, we found no evidence for pronounced performance deficits in people hypothesized to be at risk for bipolar and unipolar affective disorder. Nevertheless, in line with other studies reporting sustained attention deficit in bipolar disorder (e.g., Clark et al., 2002; Wilder-Willis et al., 2001), we found that the group being at risk for bipolar disorder was less able to discriminate between targets and nontargets in the CPT but only when they were schizotypal as well. Differences in error rates on the d2 Test were related to risk status but disappeared when controlling for general intelligence. The only remaining difference—even after controlling for current mood symptoms and general intellectual functioning—was that people thought to be at risk for unipolar depression actually had a low reaction criterion, i.e., reacted rather too fast and often than missing targets, i.e., they did not adopt a conservative strategy.

Because of prior research, we did not expect the group with high rigidity scores, and therefore putatively at risk for unipolar depression, to show marked deficits in neuropsychological deficits as long as general intellectual functioning and current symptoms are controlled for (e.g., Fossati et al., 2004; Liu et al., 2002). In contrast to Liu et al. (2002) and to Erlenmeyer-Kimling and Comblatt (1992), people hypothesized to be at risk for depression had a risky reaction criterion (ln beta) that implies faster reactions and more false alarms without showing a superior performance. In the selective attention task, a similar trend for these individuals was observed that, however, disappeared after controlling for mood and intelligence. Keeping in mind that we defined risk using the Rigidity Scale (von Zerssen et al., 1988) based on the Typus Melancholicus, one could also have expected that they would avoid any error if possible. Looking at their performance, it resembles more that of persons with obsessive–compulsive disorder (e.g., Bannon et al., 2002). No longitudinal study has yet investigated what specific problems people are prone to if they have high scores on rigidity; therefore, the assumption that rigidity is a vulnerability marker for depression is based on theoretical models and research done with relatives of patients (e.g., Lauer et al., 1997; Maier et al., 1992; von Zerssen, 1996).

Switching to the vulnerability for bipolar disorder, on the one hand, we find support for the idea that deficits in sustained attention is reported for remitted patients (e.g., Clark et al., 2002; Wilder-Willis et al., 2001), on the other hand, we found support for the hypothesis that such deficits are not generally present in people at risk for bipolar disorder. First of all, there is evidence that neuropsychological abnormalities in bipolar disorder are associated with the number of episodes, severity, and duration of illness (e.g., Clark et al., 2002; Martinez-Aran et al., 2004; van Gorp et al., 1998) and therefore might indicate a neurodegenerative effect of the disorder. Second, the reduced sensitivity on the CPT was found more pronounced in those hypomanic persons who also scored high on Magical Ideation, i.e., a schizotypy measure. This fits results showing that neuropsychological impairments are present in affective disorder especially when psychotic symptoms are present in acute phases as well (e.g., Albus et al., 1996; Liu et al., 2002; Mojtabai et al., 2000). Similarly, Tabares-Seisdedos et al. (2003) found that, in general, the performance of schizophrenic and bipolar patients did not differ, but it mattered if
there was a positive or negative family history of psychosis regardless of the specific diagnosis. Our results suggest that people at risk for bipolar disorder might differ in their risk for psychosis, and that the latter is correlated with neuropsychological abnormalities.

Before drawing final conclusions, one should keep in mind the following limitations of the study. First, we only considered two aspects of attention and did not consider other cognitive tasks, e.g., memory or executive functions. Pursuing a multimethod approach would have allowed determining the specificity of our results. Furthermore, referring to the discussion if schizophrenia and bipolar disorder are more distinct conditions or similar to each other, the inclusion of an additional purely psychosis-prone group would have been better than just controlling for schizotypal traits. Finally, vulnerability for affective disorders was only defined psychometrically using the HPS and Rigidity Scale. Although there is evidence that these traits are associated with risk for affective disorders (e.g., Kwapil et al., 2000; Maier et al., 1992; Meyer, 2002a), we do not know if the results obtained can be generalized to the area of biological-genetic risk. However, on the other hand, the main advantage of a vulnerability indicator such as the Hypomanic Personality Scale is that the definition of the risk is not based on the biological relationship with an index patient so that large-scale studies are possible. Furthermore, we do not know yet if obvious familial or non-familial forms of bipolar disorders might even involve different etiological pathways (Moorhead and Scott, 2000). Our sample consisted of healthy students scoring high on potential risk indicators for affective disorders. Although prospective follow-up assessments are planned, we do not know yet who will develop affective disorders and who will not, so that the definite risk status cannot be established yet. If there is a cognitive prodrôme for affective disorders, it was not evident in this sample. To enhance specificity, it seems worth to take into account different combinations of risk factors because our results make it evident that there is no overall deficit in attentional functioning in people hypothesized to be at risk for bipolar disorder, but there is evidence for problems with sustained attention in schizotypal hypomanic individuals.

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